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Summary

# EFFECTS OF GUT NLRP3 INHIBITION IN A PARKINSON'S DISEASE MURINE MODEL

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**Oral administration of a gut-specific NLRP3 inflammasome inhibitor improved motor function, reduced neuroinflammation, and prevented alpha-synuclein accumulation in a rotenone-induced mouse model of Parkinson's disease.**

## KEY POINTS

- In rotenone-treated mice, the non-absorbable NLRP3 inhibitor INF176 (1.25 mg/kg for 5 weeks) restored motor function in rotarod testing and prevented alpha-synuclein accumulation in the midbrain while reducing expression of tyrosine hydroxidase loss.
- INF176 treatment reduced neuroinflammation markers including microglial Iba-1 expression and NLRP3 inflammasome components (ASC, caspase 1, IL-1β) and restored gut barrier integrity in rotenone-exposed mice.
- Rotenone caused defective autophagy at the central nervous system level with decreased p-mTOR and LC3B-II expression, and these autophagic parameters were restored by INF176 treatment.
- The authors suggest that targeting intestinal NLRP3 inflammasome may represent a therapeutic approach for Parkinson's disease neurodegeneration by modulating the gut-brain axis.

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